Gene Summary for lbp-7

Specify a gene using a gene name (unc-26), a predicted gene id (R13A5.9), or a protein ID (CE02711) lbp-7

[identification] [location] [function] [expression] [gene ontology] [alleles] [similarities] [reagents]

Identification

IDs:

Main name	Sequence name	Other name(s)	W
lbp-7 - (*Lipid Binding Protein*) via person evidence: John Plenefisch	T22G5.2	NM_074039 (inferred automatically) 5O359 (inferred automatically)	WBC

Concise Description: lbp-7 encodes a predicted intracellular fatty acid binding protein (i most similar to the vertebrate muscle and heart FABPs; by homol predicted to function as an intracellular transporter for small hydr molecules such as lipids and steroid hormones; as loss of lbp-7 a large-scale RNAi screens does not result in any obvious abnorma precise role of LBP-7 in C. elegans development and/or behavior known. [details]

NCBI KOGs*: Fatty acid-binding protein FABP [KOG4015]; [OMpre_WH000485

Species: *Caenorhabditis elegans*

NCBI: [AceView: 5O359]

Gene model (s):

Gene Model	Status	Nucleotides (coding/transcript)	Protei
T22G5.2 1	partially confirmed by cDNA(s)	414/559 bp	WP:CE1:

⊞ **Footnotes**

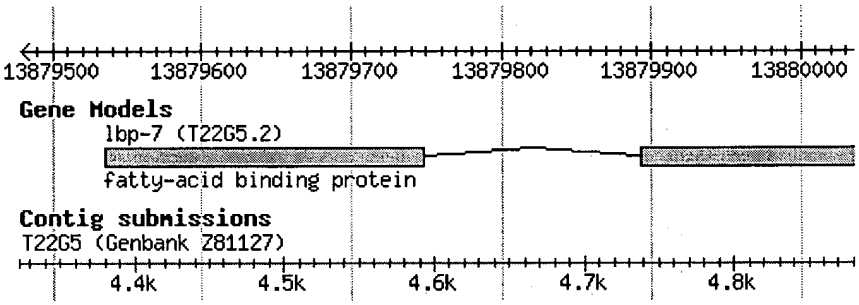
⊞ History

Location

Interpolated Genetic Position: V:5.72

Genomic Position:

Genomic Environs:



Function

Pre-WormBase information:	Definitions of abbreviations used in the text.
Phenotypes:	⊞ **phenotypes via RNAi**
Interactions:	⊞ **Interactions**
Microarray Expression Data:	SMD_T22G5.2 192878_at Aff_T22G5.2 cea2.i.45011
Expression Cluster:	cgc4386_cluster_1_3 cgc4489_group_1 WBPaper00024393:strongly_regulated_dau WBPaper00025040:N2_Expressed_Genes WBPaper00026980:intestine_enriched WBPaper00029437:rde-4_upregulated [cgc5976]:class_2 [cgc6390]:Cluster_C
Microarray "topology map" position:	Mountain [see Kim et al. Science (2001)]
Regulation on Expression Level:	nhr-49 [details]
Protein domains:	Calycin Lipocalin-related protein and Bos/Can/Equ a Cytosolic fatty-acid binding Lipocalin / cytosolic fatty-acid binding protei [more ...]

Gene Ontology

lbp-7 gene ontology summary

Biological process	determination of adult life span	(IMP) via Phenotype: 0000039; 00063219
	transport	(IEA) via InterPro

	Molecular function	binding	(IEA) via InterPro
		lipid binding	(IEA) via InterPro
		transporter activity	(IEA) via InterPro

Alleles

Homology

Ortholog(s): *Caenorhabditis briggsae:* Cbr-lbp-7 [syntenic alignment] (via a OrthoMCL; TreeFam; Inparanoid_6; OMA; WormBase-Compa

Caenorhabditis briggsae: CBG11456 [syntenic alignment] (via WormBase-Compara)

Caenorhabditis remanei: Cre-lbp-7 [syntenic alignment] (via a WormBase-Compara; Inparanoid_6; TreeFam)

InParanoid group(s): ⊞ **2 InParanoid groups for lbp-7**

*Read more about **InParanoid** on the WormBaseWiki*

TreeFam: TreeFam ID: TF316894

⊞ **Treefam image**

Phylogenetic trees provided by the Treefam project.

Similarities

Best BLASTP matches to longest protein product (full list):

(Show alignments):

Species	Hit	Description
C. remanei	RP03217 *Note: **C. remanei** predictions are based on an early assembly of the genome. Predictions subject to possibly dramatic revision pending final assembly. Sequences available on the WormBase FTP site.*	
C. briggsae	BP:CBP02793	gene CBG11457
C. elegans	WP:CE41159	retinol binding protein
S. tridecemlineatus	SW:Q99P61	Fatty acid-binding protein, heart (H-FABP) (Heart-type fatty acid-

			sbinding protein).
	C. hircus	TR:Q6S4N9	Fatty acid-binding protein.
	H. sapiens	ENSEMBL:ENSP00000362817	Fatty acid-binding protein, heart
	D. melanogaster	FLYBASE:CG6783-PB	Flybase gene name is CG6783-PB

Reagents	**ORFeome Project primers & sequences:**	mv_T22G5.2 OSTF158E7_1 OSTR158E7_1		
	Primer pairs:	sjj_T22G5.2 cenix:44-g12		
	Microarray probes:	192878_at [Affymetrix]	cea2.i.45011 [GSC]	A_12_ [Agilen
	SAGE Tags:	SAGE:gaactttgat SAGE:aaggaaatcg		
	Matching cDNAs:	OSTF158E7_1 OSTR158E7_1		
Bibliography	**Total citations for lbp-7:** 4 [view all]			

Submit your data or corrections

Send corrections to WormBase via email
Submit additional annotations on lbp-7 on the WormBase Wiki

Display Full Report Show 20 Send to

All: 1 Current Only: 1 **Genes Genomes: 1** SNP GeneView: 0

1: lbp-7 Lipid Binding Protein [*Caenorhabditis elegans*]

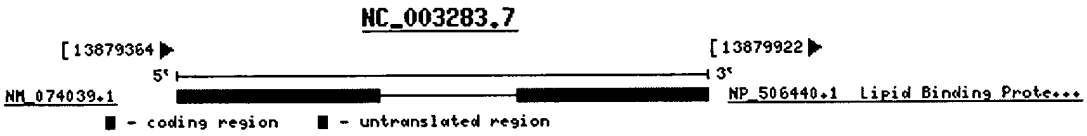
GeneID: 191701 updated 13-Dec-2007

Summary

Gene name	lbp-7
Gene description	Lipid Binding Protein
Primary source	WormBase:WBGene00002259
Locus tag	T22G5.2
Gene type	protein coding
RefSeq status	Reviewed
Organism	*Caenorhabditis elegans (strain: Bristol N2)*
Lineage	*Eukaryota; Metazoa; Nematoda; Chromadorea; Rhabditida; Rhabditoidea; Rhabditidae; Peloderinae; Caenorhabditis*

Genomic regions, transcripts, and products

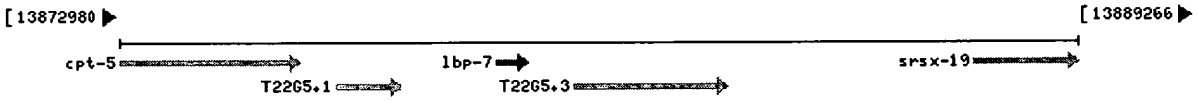
Go to reference sequence details



Genomic context

chromosome: V

See lbp-7 in MapViewer



Bibliography

Related Articles in PubMed

PubMed links

GeneRIFs: Gene References Into Function

Interactions

Description .....

Product	Interactant	Other Gene	Complex	Source	Pubs
Two-hybrid					
BioGRID:56199	BioGRID:43789	W02G9.3		BioGRID	PubMed

General gene information

GeneOntology

Provided by WormBase

Function	Evidence
binding PubMed 12520011,12654719	IEA
lipid binding PubMed 12520011,12654719	IEA
transporter activity PubMed 12520011,12654719	IEA

Process	Evidence
determination of adult life span PubMed 12845331	IMP
transport PubMed 12520011,12654719	IEA

General protein information

Names

Lipid Binding Protein family member (lbp-7)

NCBI Reference Sequences (RefSeq)

mRNA and Protein(s)

1. **NM_074039.1→NP_506440.1 Lipid Binding Protein family member (lbp-7) [Caenorhabditis elegans]**

UniProtKB/Swiss-Prot | O02323

Conserved Domains (1) summary

pfam00061 **Location:9–123** **Blast Score:119**	Lipocalin; Lipocalin / cytosolic fatty-acid binding protein family. Lipocalins are transporters for small hydrophobic molecules, such as lipids, steroid hormones, bilins, and retinoids. The family also encompasses the enzyme prostaglandin D synthase (EC:5.3.99.2).

Related Sequences

Nucleotide		Protein
Genomic	Z81127.1	CAB03387.1

Protein Accession	Links
O02323.1	GenPept

Additional Links

- UniGene Cel.2988

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AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

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=> s LBP 7 or lbp7
L1 4 LBP 7 OR LBP7

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 4 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L2 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2006:681667 BIOSIS <<LOGINID::20080104>>
DN PREV200600684105
TI Changes in the flexion relaxation response following an exercise intervention.
AU Marshall, Paul [Reprint Author]; Murphy, Bernadette
CS Univ Auckland, Dept Sport and Exercise Sci, Tamaki Campus,Private Bag 92019, Auckland, New Zealand
p.marshall@auckland.ac.nz
SO Spine, (NOV 1 2006) Vol. 31, No. 23, pp. E877-E883.
CODEN: SPINDD. ISSN: 0362-2436.
DT Article
LA English
ED Entered STN: 6 Dec 2006
Last Updated on STN: 6 Dec 2006
AB Study Design. Pre and post 12-week training study using surface electromyography to measure the flexion relaxation response.Objective. To evaluate whether the active or passive phases of the flexion relaxation measurement changes following an exercise intervention in patients with low back pain.Summary of Background Data. Impaired neuromuscular activation is an area of specific interest in patients with chronic nonspecific low back pain (LBP). The flexion relaxation phenomenon is commonly measured in LBP patients; however, there is insufficient evidence about the changes in this measure following an intervention.Methods. Fifteen subjects with chronic ***LBP*** (***7*** females, 8 males) performed a 12-week training intervention. The main outcome measures were the Oswestry disability index, visual analog scale, and flexion relaxation response analyzed by the raw electromyograph (EMG) signal, the relative EMG signal, and the flexion relaxation ratio.Results. Disability and pain scores improved significantly after the 12-week intervention. There were no changes in the active components of the flexion relaxation measurement but an approximate 67% decrease in the amount of activity measured during the relaxation phase at full trunk flexion.Conclusion. The data suggest that afferent feedback changes may be explaining why there is improved electrical relaxation following an exercise intervention.

L2 ANSWER 2 OF 4 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
AN 2006180752 EMBASE <<LOGINID::20080104>>
TI Plantar Infrared Thermography Measurements and Low Back Pain Intensity.
AU Zaproudina N.; Ming Z.; Hanninen O.O.P.
CS Dr. N. Zaproudina, Department of Physiology, University of Kuopio, Kuopio, Finland. nina.zaproudina@uku.fi
SO Journal of Manipulative and Physiological Therapeutics, (Mar 2006) Vol. 29, No. 3, pp. 219-223.
Refs: 19
ISSN: 0161-4754 CODEN: JMPTDE
PUI S 0161-4754(06)00025-X
CY United States
DT Journal; Article
FS 019 Rehabilitation and Physical Medicine
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 6 Jun 2006
Last Updated on STN: 6 Jun 2006
AB Objective: To study the skin temperature disorders in low back pain (LBP) patients compared with reference persons without LBP and to evaluate the relationship between pain intensity and other clinical signs and temperature abnormalities. Methods: Sixty-five patients with unilateral chronic LBP with or without referred nonradicular leg pain (29 men and 36 women; age range, 30-51 years) and 20 reference persons without ***LBP*** (***7*** men and 13 women; age range, 30-49 years) participated in this study. The pain level was recorded by the use of a visual analog scale (0-100). Questionnaires and a series of spinal mobility tests (the modified Schober, straight leg-raising test, finger-floor distance, side bending) were used. Thermographic images of the low back area and legs (anterior, lateral, and posterior surfaces and the plantar surfaces of feet) were taken with an infrared video camera. Results: The temperature changes in the plantar surface correlated with LBP intensity. The pain levels differed in the groups with the different types of temperature changes. There were significant lower extremity regional skin temperature alterations (at least 1 regional interside difference more than 0.3.degree.C) in most cases both in LBP patients and in reference persons, but plantar interside temperature difference was significantly higher in LBP patients. Conclusion: Temperature changes of the plantar surface seem to be connected with LBP intensity. Temperature

measurements may be useful as an adjunctive physiological test in the evaluation and documentation of autonomic dysfunction in LBP patients. . .COPYRGT. 2006 National University of Health Sciences.

L2 ANSWER 3 OF 4 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
AN 2006537243 EMBASE <<LOGINID::20080104>>
TI Changes in the flexion relaxation response following an exercise intervention.
AU Marshall P.; Murphy B.
CS P. Marshall, Department of Sport and Exercise Science, University of Auckland, Tamaki Campus, Private Bag 92019, Auckland, New Zealand. p.marshall@auckland.ac.nz
SO Spine, (Nov 2006) Vol. 31, No. 23, pp. E877-E883.
Refs: 45
ISSN: 0362-2436 E-ISSN: 1528-1159 CODEN: SPINDD
PUI 00007632006110100025
CY United States
DT Journal; Article
FS 033 Orthopedic Surgery
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 16 Nov 2006
Last Updated on STN: 16 Nov 2006
AB STUDY DESIGN. Pre and post 12-week training study using surface electromyography to measure the flexion relaxation response. OBJECTIVE. To evaluate whether the active or passive phases of the flexion relaxation measurement changes following an exercise intervention in patients with low back pain. SUMMARY OF BACKGROUND DATA. Impaired neuromuscular activation is an area of specific interest in patients with chronic nonspecific low back pain (LBP). The flexion relaxation phenomenon is commonly measured in LBP patients; however, there is insufficient evidence about the changes in this measure following an intervention. METHODS. Fifteen subjects with chronic ***LBP*** (***7*** females, 8 males) performed a 12-week training intervention. The main outcome measures were the Oswestry disability index, visual analog scale, and flexion relaxation response analyzed by the raw electromyograph (EMG) signal, the relative EMG signal, and the flexion relaxation ratio. RESULTS. Disability and pain scores improved significantly after the 12-week intervention. There were no changes in the active components of the flexion relaxation measurement but an approximate 67% decrease in the amount of activity measured during the relaxation phase at full trunk flexion. CONCLUSION. The data suggest that afferent feedback changes may be explaining why there is improved electrical relaxation following an exercise intervention. .COPYRGT. 2006 Lippincott Williams & Wilkins, Inc.

L2 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1993:532618 CAPLUS <<LOGINID::20080104>>
DN 119:132618
TI Determination of pig apolipoprotein B genotype by gene amplification and restriction fragment length polymorphism analysis
AU Kaiser, M. E.; Nevin, D. N.; Sturley, S. L.; Purtell, C.; Attie, A. D.
CS Dep. Comp. Biosci., Univ. Wisconsin, Madison, WI, 53706, USA
SO Animal Genetics (1993), 24(2), 117-20
CODEN: ANGEE3; ISSN: 0268-9146
DT Journal
LA English
AB Sequence differences within the pig apolipoprotein B (apoB) gene can be used to identify rapidly four of eight known pig apoB alleles, designated LPB1-LBP8. The use of gene amplification, followed by endonuclease digestion and agarose gel electrophoresis, to discern size and restriction site differences is described. LBP5, a common allele assocd. with reduced low d. lipoprotein clearance and hypercholesterolemia in pigs, is identified by a 283-bp insertion in intron 28. LBP3 and ***LBP7*** are distinguished by a unique HindIII site; LBP8 shares a unique HincII site with LBP5. This method facilitates identification of the apoB genotype of pigs used in lipoprotein research and allows for further investigation into the assocn. of particular apoB alleles with lipoprotein metab. abnormalities.

=> s T22G5.2
L3 1 T22G5.2

=> d bib abs

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2003:6104 CAPLUS <<LOGINID::20080104>>
DN 138:67843
TI Methods of assaying genes of C. elegans involved in adult lifespan regulation and uses for gene therapy
IN Kenyon, Cynthia; Apfeld, Javier; Dillin, Andrew; Garigan, Delia; Hsu, Ao-Lin A.; Lehrer-Graiwer, Josh; Murphy, Coleen
PA The Regents of the University of California, USA
SO PCT Int. Appl., 112 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003000861	A2	20030103	WO 2002-US20247	20020624
WO 2003000861	A9	20030410		
WO 2003000861	A3	20030710		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2451247	A1	20030103	CA 2002-2451247	20020624
AU 2002350597	A1	20030108	AU 2002-350597	20020624
US 2003190312	A1	20031009	US 2002-179766	20020624
EP 1406489	A2	20040414	EP 2002-752095	20020624

R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR

US 2006162002	A1	20060720	US 2005-282262	20051118

PRAI US 2001-300577P	P	20010622
US 2001-301052P	P	20010625
US 2002-373975P	P	20020418
US 2002-179766	A1	20020624
WO 2002-US20247	W	20020624

AB The present invention is directed to methods of assaying for genes, gene products, and genes in pathways controlled by such genes and gene products, using RNAi and microarray anal., that regulate lifespan (e.g., extend or truncate adult lifespan) in eukaryotes such as invertebrates (e.g., C. elegans), plants, and mammals, e.g., humans. For example, the present invention is directed to genes encoding components of the mitochondrial respiratory chain and genes encoding glycolysis enzymes, which are involved in lifespan regulation, and genes and gene products in pathways controlled by such genes. Other genes and gene products identified as regulating aging and aging pathways include a gene encoding a GTPase; a transcriptional activator; novel genes: llw-1, llw-2, llw-3, and llw-4; genes encoding cytochrome P 450 proteins (involved in steroid biosynthesis); a melatonin synthesis gene; genes encoding insulin and insulin-like peptides; genes encoding heat shock factors; genes encoding catalases; stress-response genes; and metabolic genes. The invention further relates to methods for identifying and using agents, including small mol. chem. compns., antibodies, antisense nucleic acids, and ribozymes, that regulate, e.g., enhance, adult lifespan via modulation of aging assocd. proteins; as well as to the use of expression profiles, markers, and compns. in diagnosis and therapy related to lifespan extension, life expectancy, and aging. The present invention also relates to gene therapy involving lifespan assocd. genes.

=> s age or aging or lifespan
L4 2626198 AGE OR AGING OR LIFESPAN

=> s l4 and regula?
L5 149161 L4 AND REGULA?

=> s l5 and review
L6 13428 L5 AND REVIEW

=> d bib abs 1-10

L6 ANSWER 1 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:49198 BIOSIS <<LOGINID::20080104>>
DN PREV200800045245
TI Kinetics, role and therapeutic implications of endogenous soluble form of receptor for advanced glycation end products (sRAGE) in diabetes.
AU Yamagishi, Sho-ichi [Reprint Author]; Matsui, Takanori; Nakamura, Kazuo
CS Kurume Univ, Sch Med, Dept Med, Div Cardiovasc Med, Kurume, Fukuoka 8300011, Japan
shoichi@med.kurume-u.ac.jp
SO Current Drug Targets, (OCT 2007) Vol. 8, No. 10, pp. 1138-1143.
ISSN: 1389-4501.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 4 Jan 2008
Last Updated on STN: 4 Jan 2008
AB Reducing sugars can react non-enzymatically with amino groups of protein to form Amadori products. These early glycation products undergo further complex reaction such as rearrangement, dehydration, and condensation to become irreversibly cross-linked, heterogeneous fluorescent derivatives, termed advanced glycation end products (AGEs). The formation and accumulation of AGEs have been known to progress at an accelerated rate in diabetes. There is a growing body of evidence that AGEs and their receptor (RAGE) axis is implicated in the pathogenesis of diabetic vascular complications. Indeed, the engagement of RAGE with AGEs is shown to elicit oxidative stress generation and subsequently evoke inflammatory responses in various types of cells, thus playing an important role in the development and progression of diabetic micro- and macroangiopathy. Moreover, administration of a recombinant soluble form of RAGE (sRAGE), has been shown to suppress the development of accelerated atherosclerosis

in diabetic apolipoprotein E-null mice. These observations suggest that exogenously administered sRAGE may capture and eliminate circulating AGEs, thus protecting against the AGEs-elicited tissue damage by acting as a decoy receptor. Recently, endogenous sRAGE has been identified in humans. However, there is few comprehensive ***review*** about the ***regulation*** and role of endogenous sRAGE in diabetes. In the former part of this paper, we ***review*** the role of the ***AGE*** -RAGE system in the pathogenesis of diabetic vascular complications. Then we summarize in the latter part of this ***review*** the kinetics and pathophysiological role of endogenous sRAGE in diabetes. We also discuss the possibility that endogenous sRAGE may be a therapeutic target for the prevention of diabetic vascular complications.

L6 ANSWER 2 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:46781 BIOSIS <<LOGINID::20080104>>
DN PREV200800045726
TI Quality-of-life in elderly patients with cancer: A short ***review*** .
AU Wedding, Ulrich [Reprint Author]; Pientka, Ludger; Hoeffken, Klaus
CS Univ Jena, Dept Haematol and Med Oncol, Internal Med Clin 2, Erlanger Allee 101, D-07747 Jena, Germany
ulrich.wedding@med.uni-jena.de
SO European Journal of Cancer, (OCT 2007) Vol. 43, No. 15, pp. 2203-2210. CODEN: EJCAEL. ISSN: 0959-8049.
DT Article
LA English
ED Entered STN: 4 Jan 2008
Last Updated on STN: 4 Jan 2008
AB Background: Prolongation of survival and maintenance or improvement of health-related quality-of-life (HRQoL) are the two important goals within the treatment of individual patients. Due to the severity of symptoms and the toxicity of treatment, HRQoL has become a major area of concern when treating cancer patients in general and elderly patients in particular.Patients and methods: We present a literature ***review*** of HRQoL aspects in elderly patients with cancer and especially address the topic whether impairments in the different tools of a comprehensive geriatric assessment (CGA) are associated with decreased HRQoL in elderly cancer patients.Results: Elderly cancer patients tend to weight their HRQoL as more important than gain in survival, when compared to younger patients. An ***age*** -dependent decrease in different scales of HRQoL is reported in patients and normative samples. HRQoL is also a predictor of survival. The variation of HRQoL can be used in trials comparing different treatment options. In individual patients, ***regular*** measurement of HRQoL aims to improve patients-centred care. ***Age*** related impairments of different areas of CGA are associated with decreased HRQoL in elderly cancer patients.Conclusions: HRQoL is an important outcome with elderly cancer patients and should be assessed ***regularly*** and thoroughly. (C) 2007 Elsevier Ltd. All rights reserved.

L6 ANSWER 3 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:42460 BIOSIS <<LOGINID::20080104>>
DN PREV200800036414
TI Telomere length profiles in humans - All ends are not equal.
AU Gilson, Eric; Londono-Vallejo, Arturo [Reprint Author]
CS UMR, Inst Curie, Telomere and Canc Lab, Rue Ulm, F-75248 Paris, France
Arturo.Londono@curie.fr
SO Cell Cycle, (OCT 15 2007) Vol. 6, No. 20, pp. 2486-2494. ISSN: 1538-4101. E-ISSN: 1551-4005.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 27 Dec 2007
Last Updated on STN: 27 Dec 2007
AB Telomere length is an important parameter of telomere function since it determines number of aspects controlling chromosome stability and cell division. Since telomeres shorten with ***age*** in humans and premature ***aging*** syndromes are often associated with the presence of short telomeres, it has been proposed that telomere length is also an important parameter for organismal ***aging*** . How mean telomere lengths are determined in humans remains puzzling, but it is clear that genetic and epigenetic factors appear to be of great importance. Experimental evidence obtained from many different organisms has provided the basis for a widely accepted counting mechanism based on a negative feedback loop for telomerase activity at the level of individual telomeres. In addition, recent studies in both normal and pathological contexts point to the existence of chromosome-specific mechanisms of telomere length ***regulation*** determining a telomere length profile, which is inherited and maintained throughout life. In this ***review*** , we recapitulate the available data, propose a synthetic view of telomere length control mechanisms in humans and suggest new approaches to test current hypotheses.

L6 ANSWER 4 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:31851 BIOSIS <<LOGINID::20080104>>
DN PREV200800029157
TI Adipokine gene expression in brain and pituitary gland.
AU Wilkinson, Michael [Reprint Author]; Brown, Russell; Imran, Syed A.; Ur, Ehud
CS IWK Hlth Ctr, Dept Obstet and Gynaecol, 5980 Univ Ave,PO Box 9700, Halifax, NS B3K 6R8, Canada
mwilk@dal.ca
SO Neuroendocrinology, (2007) Vol. 86, No. 3, pp. 191-209. CODEN: NUNDAJ. ISSN: 0028-3835.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 19 Dec 2007
Last Updated on STN: 19 Dec 2007
AB The brain has been recognized as a prominent site of peptide biosynthesis for more than 30 years, and many neuropeptides are now known to be common to gut and brain. With these precedents in mind it is remarkable that adiposederived peptides like leptin have attracted minimal attention as brain-derived putative neuromodulators of energy balance. This ***review*** outlines the evidence that several adipose-specific genes are also expressed in the central nervous system and pituitary gland. We, and others, confirmed that the genes for leptin, resistin, adiponectin, FIAF (fasting-induced adipose factor) and adiponutrin are expressed and ***regulated*** in these tissues. For example, leptin mRNA was readily detectable in human, rat, sheep and pig brain, but not in the mouse. Leptin expression in rat brain and pituitary was ***regulated*** through development, by food restriction, and following traumatic brain injury. In contrast, hypothalamic resistin mRNA was unaffected by ***age*** or by fasting, but was significantly depleted by food restriction in mouse pituitary gland. Similar results were seen in the ob/ob mouse, and we noted a marked reduction in resistin-positive hypothalamic nerve fibres. Resistin and fiaf mRNA were also upregulated in hypoxic/ischaemic mouse brain. Our studies on the ***regulation*** of neuronal adipokines were greatly aided by the availability of clonal hypothalamic neuronal cell lines. One of these, N-1, expresses both rstn and fiaf together with several other neuropeptides and receptors involved in energy homeostasis. Selective silencing of rstn revealed an autocrine/ paracrine ***regulatory*** system, mediated through socs-3 expression that may influence the feedback effects of insulin and leptin in vivo. A similar convergence of signals in the pituitary gland could also influence anterior pituitary hormone secretion. In conclusion, the evidence is suggestive that brain and pituitary-derived adipokines represent a local ***regulatory*** circuit that may fine tune the feedback effects of adipose hormones in the control of energy balance. Copyright (C) 2007 S. Karger AG, Basel

L6 ANSWER 5 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:27148 BIOSIS <<LOGINID::20080104>>
DN PREV200800017125
TI Methionine sulfoxide reductases: selenoprotein forms and roles in antioxidant protein repair in mammals.
AU Kim, Hwa-Young [Reprint Author]; Gladyshev, Vadim N.
CS Yeungnam Univ, Coll Med, Aging Associated Vasc Dis Res Ctr, Dept Biochem and Mol Biol, Taegu 705717, South Korea
hykim@ynu.ac.kr; vgladyshev1@unl.edu
SO Biochemical Journal, (NOV 1 2007) Vol. 407, No. Part 3, pp. 321-329. ISSN: 0264-6021. E-ISSN: 1470-8728.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 19 Dec 2007
Last Updated on STN: 19 Dec 2007
AB Msrs (methionine sulfoxide reductases), MsrA and MsrB, are repair enzymes that reduce methionine sulfoxide residues in oxidatively damaged proteins to methionine residues in a stereospecific manner. These enzymes protect cells from oxidative stress and have been implicated in delaying the ***aging*** process and progression of neurodegenerative diseases. In recent years, significant efforts have been made to explore the catalytic properties and physiological functions of these enzymes. In the current ***review*** , we present recent progress in this area, with the focus on mammalian MsrA and MsrBs including their roles in disease, evolution and function of selenoprotein forms of MsrA and MsrB, and the biochemistry of these enzymes.

L6 ANSWER 6 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:25935 BIOSIS <<LOGINID::20080104>>
DN PREV200800029314
TI Association between genetic variants in sortilin-related receptor 1 (SORL1) and Alzheimer's disease in adults with Down syndrome.
AU Lee, Joseph. H.; Chulikavit, Maruit; Pang, Deborah; Zigman, Warren B.; Silverman, Wayne; Schupf, Nicole [Reprint Author]
CS Taub Inst Res Alzheimers Dis and Aging Brain, PO Box 16,630 W 168th St, New York, NY 10032 USA
ns24@columbia.edu
SO Neuroscience Letters, (SEP 25 2007) Vol. 425, No. 2, pp. 105-109. CODEN: NELED5. ISSN: 0304-3940.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 19 Dec 2007
Last Updated on STN: 19 Dec 2007
AB Recent reports have suggested that variants in the sortilin-related

receptor gene (SORL1) increase the risk of late onset Alzheimer's disease (AD) in Northern European, Hispanic, African-American and Isreali-Arab populations. SORL1 directs trafficking of amyloid precursor protein (APP) and under-expression of SORL1 may lead to over-expression of P amyloid peptides. Adults with Down syndrome (DS) over-express APP and have early onset and high risk for AD. We investigated the relation of seven variants in the gene for SORL1 to ***age*** at onset and risk for AD among 208 adults with DS, 45-70 years of ***age*** at baseline. Participants were ascertained through the New York State developmental disability service system and followed at 18-month intervals. Information from cognitive assessments, caregiver interviews, medical record ***review*** and neurological examination was used to establish the diagnosis of dementia. Homozygosity for the minor T allele in rs556349 and for the minor C allele in rs536360 was associated with later ***age*** at onset and reduced risk of AD (HR = 0.26, 95% CI: 0.08-0.86; and HR = 0.40, 95% CI: 0.16-0.98, respectively). Mean ***age*** at onset was approximately four years later in individuals who were homozygous for those alleles compared with those who had at least one major allele. These findings indicate a modest association of variants in SORL1 with AD. In addition, we did not observe the same alleles to be associated with AD compared with earlier studies, suggesting that these SNPs are in linkage disequilibrium (LD) with the putative functional variants or that expression of the SORL1 gene and hence its interaction with APP might be modified by the extremely high levels of APP characteristic of Down syndrome. Thus, further studies are needed to identify functional variants that influence risk for AD in this uniquely vulnerable population. (c) 2007 Elsevier Ireland Ltd. All rights reserved.

L6 ANSWER 7 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:24407 BIOSIS <<LOGINID::20080104>>
DN PREV200800031491
TI Fluid and electrolyte homeostasis in healthy subjects during prolonged hypokinesia.
AU Tsiamis, C. B. [Reprint Author]
CS Odos Kerasundos 2-4, GR-16232 Athens, Greece
cbtsiamis@in.gr
SO Trace Elements and Electrolytes, (2007) Vol. 24, No. 4, pp. 193-201.
ISSN: 0946-2104.
DT Article
LA English
ED Entered STN: 19 Dec 2007
Last Updated on STN: 19 Dec 2007
AB Hypokinesia (diminished movement) is a factor of catabolism induction which may be developed, whenever there is a significant reduction of physical load on skeletal muscle system. Hypokinesia (HK) may occur due to ***age*** , disease, disability and sedentary living and working conditions. Among the effects of HK, the changes in fluid and electrolytes (sodium, chloride, potassium, phosphorus, calcium and magnesium) homeostasis and in particular in fluid and electrolyte deposition (ability of the body to use electrolytes and fluid after all fluid and electrolyte loss have been established) have been of the greatest interest. This is due to the higher fluid and electrolyte loss with higher than lower fluid and electrolyte depletion and the recent interest on fluid and electrolyte homeostasis and fluid and electrolyte deposition under sedentary living and working conditions. Stable volume, osmotic concentration and electrolyte composition of internal fluid is one of the mandatory prerequisites for humans to maintain adequate physiological and biochemical condition and a highly efficient physical state. However, physiological and biochemical systems that ***regulate*** the concentration of each electrolyte in blood and other endogenous fluids, and the balance between the consumption and elimination of electrolytes from the body and thus total electrolyte content of the body are drastically affected. At present some information has been accumulated about the effect of prolonged HK on fluid and electrolyte homeostasis and fluid and electrolyte loss with fluid and electrolyte depletion. To this end, the aim of this ***review*** was to report the findings on the fluid and electrolyte homeostasis and fluid-electrolyte loss with whole body fluid and electrolyte depletion.

L6 ANSWER 8 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:13833 BIOSIS <<LOGINID::20080104>>
DN PREV200800006313
TI Mood disorders and fertility in women: a critical ***review*** of the literature and implications for future research.
AU Williams, Katherine E. [Reprint Author]; Marsh, Wendy K.; Rasgon, Natalie L.
CS Stanford Univ, Dept Psychiat and Behav Sci, Stanford Ctr Neurosci Womenns Helath, Stanford, CA 94305 USA
elliew@stanford.edu
SO Human Reproduction Update, (NOV-DEC 2007) Vol. 13, No. 6, pp. 607-616.
ISSN: 1355-4786.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 12 Dec 2007
Last Updated on STN: 27 Dec 2007
AB A medline literature ***review*** of fertility and mood disorder articles published since 1980 was performed in order to critically ***review*** the literature regarding a relationship between mood disorders, fertility and infertility treatment. Previous studies suggests that mood disorders, both in the bipolar and unipolar spectrum, may be associated with decreased fertility rates. Most studies report that women seeking treatment for infertility have an increased rate of depressive symptoms and possibly major depression (none showed evaluated mood elevations). Many, but not all, studies found that depressive symptoms may decrease the success rate of fertility treatment. Treatments for infertility may independently influence mood through their effects on estrogen and progesterone, which have been shown to influence mood through their actions on serotonin. Studies are limited in scope and confounding variables are many, limiting the strength of the results. In conclusion, a range of existing studies suggests that fertility and mood disorders are related in a complex way. Future studies should use clinical interviews and standardized and validated measures to confirm the diagnosis of mood disorders and control for the variables of medication treatment, desire for children, frequency of sexual intercourse, ***age*** , FSH levels, menstrual cycle ***regularity*** in assessing an interrelationship between mood disorders and fertility.

L6 ANSWER 9 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:12030 BIOSIS <<LOGINID::20080104>>
DN PREV200800005089
TI Long-chain polyunsaturated fatty acids and the ***regulation*** of bone metabolism.
AU Poulsen, Raewyn C. [Reprint Author]; Moughan, Paul J.; Kruger, Marlena C.
CS Inst Food Nutr and Human Hlth, Private Bag 11-222, Palmerston North New Zealand
R.C.Poulsen@massey.ac.nz
SO Experimental Biology and Medicine (Maywood), (NOV 2007) Vol. 232, No. 10, pp. 1275-1288.
ISSN: 1535-3702.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 12 Dec 2007
Last Updated on STN: 12 Dec 2007
AB The role of prostaglandin E2 (PGE2) in the ***regulation*** of bone remodeling is well established. There is increasing evidence that various long-chain polyunsaturated fatty acids (LCPUFAs), as well as nonprostanoid LCPUFA metabolites, also have critical roles in ***regulating*** bone metabolism and may have therapeutic potential in the management of postmenopausal osteoporosis. Although only the 18-carbon precursors for the n-3 and n-6 LCPUFAs are deemed "dietary essential," the ability of the body to convert these precursor fatty acids into the more highly unsaturated 20- and 22-carbon LCPUFAs decreases with ***aging*** , menopause, and various lifestyle factors (e.g., smoking). Increasing dietary LCPUFA intake increases tissue and blood LCPUFA concentrations, as well as the concentrations of their metabolites. Modification of dietary LCPUFA content, particularly increasing the intake of n-3 LCPUFAs, has been shown to minimize the decline in bone mass caused by menopause in women and ovariectomy in animal models. This ***review*** summarizes findings from both in vivo and in vitro studies and outlines the effects of LCPUFAs and their metabolites on calcium balance, osteoblastogenesis, osteoclastogenesis, and osteoblast and osteoclast function.

L6 ANSWER 10 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:11678 BIOSIS <<LOGINID::20080104>>
DN PREV200800004301
TI Controlling mutation: intervening in evolution as a therapeutic strategy.
AU Cirz, Ryan T.; Romesberg, Floyd E. [Reprint Author]
CS Scripps Res Inst, 10550 N Torrey Pines Rd, La Jolla, CA 92037 USA
floyd@scripps.edu
SO Critical Reviews in Biochemistry and Molecular Biology, (2007) Vol. 42, No. 5, pp. 341-354.
CODEN: CRBBEJ. ISSN: 1040-9238.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 12 Dec 2007
Last Updated on STN: 12 Dec 2007
AB Mutation is the driving force behind many processes linked to human disease, including cancer, ***aging*** , and the evolution of drug resistance. Mutations have traditionally been considered the inevitable consequence of replicating large genomes with polymerases of finite fidelity. Observations over the past several decades, however, have led to a new perspective on the process of mutagenesis. It has become clear that, under some circumstances, mutagenesis is a ***regulated*** process that requires the induction of pro-mutagenic enzymes and that, at least in bacteria, this induction may facilitate evolution. Herein, we ***review*** what is known about induced mutagenesis in bacteria as well as evidence that it contributes to the evolution of antibiotic resistance. Finally, we discuss the possibility that components of induced mutation pathways might be targeted for inhibition as a novel therapeutic strategy to prevent the evolution of antibiotic resistance.

=> d his

(FILE 'HOME' ENTERED AT 18:47:57 ON 04 JAN 2008)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 18:48:06 ON 04 JAN 2008
L1 4 S LBP 7 OR LBP7
L2 4 DUP REM L1 (0 DUPLICATES REMOVED)
L3 1 S T22G5.2
L4 2626198 S AGE OR AGING OR LIFESPAN
L5 149161 S L4 AND REGULA?
L6 13428 S L5 AND REVIEW

=> s l4 (3a) regulat?
L7 5534 L4 (3A) REGULAT?

=> s l7 and review
L8 929 L7 AND REVIEW

=> d bib abs 1-10

L8 ANSWER 1 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2008:49198 BIOSIS <<LOGINID::20080104>>
DN PREV200800045245
TI Kinetics, role and therapeutic implications of endogenous soluble form of receptor for advanced glycation end products (sRAGE) in diabetes.
AU Yamagishi, Sho-ichi [Reprint Author]; Matsui, Takanori; Nakamura, Kazuo
CS Kurume Univ, Sch Med, Dept Med, Div Cardiovasc Med, Kurume, Fukuoka 8300011, Japan
shoichi@med.kurume-u.ac.jp
SO Current Drug Targets, (OCT 2007) Vol. 8, No. 10, pp. 1138-1143. ISSN: 1389-4501.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 4 Jan 2008
Last Updated on STN: 4 Jan 2008
AB , Reducing sugars can react non-enzymatically with amino groups of protein to form Amadori products. These early glycation products undergo further complex reaction such as rearrangement, dehydration, and condensation to become irreversibly cross-linked, heterogeneous fluorescent derivatives, termed advanced glycation end products (AGEs). The formation and accumulation of AGEs have been known to progress at an accelerated rate in diabetes. There is a growing body of evidence that AGEs and their receptor (RAGE) axis is implicated in the pathogenesis of diabetic vascular complications. Indeed, the engagement of RAGE with AGEs is shown to elicit oxidative stress generation and subsequently evoke inflammatory responses in various types of cells, thus playing an important role in the development and progression of diabetic micro- and macroangiopathy. Moreover, administration of a recombinant soluble form of RAGE (sRAGE), has been shown to suppress the development of accelerated atherosclerosis in diabetic apolipoprotein E-null mice. These observations suggest that exogenously administered sRAGE may capture and eliminate circulating AGEs, thus protecting against the AGEs-elicited tissue damage by acting as a decoy receptor. Recently, endogenous sRAGE has been identified in humans. However, there is few comprehensive ***review*** about the regulation and role of endogenous sRAGE in diabetes. In the former part of this paper, we ***review*** the role of the AGE-RAGE system in the pathogenesis of diabetic vascular complications. Then we summarize in the latter part of this ***review*** the kinetics and pathophysiological role of endogenous sRAGE in diabetes. We also discuss the possibility that endogenous sRAGE may be a therapeutic target for the prevention of diabetic vascular complications.

L8 ANSWER 2 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:528569 BIOSIS <<LOGINID::20080104>>
DN PREV200700519859
TI Healthy ***aging*** : ***regulation*** of the metabolome by cellular redox modulation and prooxidant signaling systems: The essential roles of superoxide anion and hydrogen peroxide.
AU Linnane, Anthony William [Reprint Author]; Kios, Michael; Vitetta, Luis
CS Ctr Mol Biol and Med, Epworth Med Ctr, 185-187 Hoddle St, Melbourne, Vic 3121, Australia
tlinnane@cmbm.com.au
SO Biogerontology, (OCT 2007) Vol. 8, No. 5, pp. 445-467. ISSN: 1389-5729.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 10 Oct 2007
Last Updated on STN: 21 Nov 2007
AB The production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) has long been proposed as leading to random deleterious modification of macromolecules with an associated progressive development of age associated systemic disease. ROS and RNS formation has been posited as a major contributor to the aging process. On the contrary, this ***review*** presents evidence that superoxide anion (and hydrogen peroxide) and nitric oxide (and peroxynitrite) constitute regulated prooxidant second messenger systems, with specific sub-cellular locales of production and are essential for normal metabolome and physiological function. The role of these second messengers in the regulation of the metabolome is discussed in terms of radical formation as an essential contributor to the physiologically normal regulation of sub-cellular bioenergy systems; proteolysis regulation; transcription activation; enzyme activation; mitochondrial DNA changes; redox regulation of metabolism and cell differentiation; the concept that orally administered small molecule antioxidant therapy is a chimera. The formation of superoxide anion/hydrogen peroxide and nitric oxide do not conditionally lead to random macromolecular damage; under normal physiological conditions their production is actually regulated consistent with their second messenger roles.

L8 ANSWER 3 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:518754 BIOSIS <<LOGINID::20080104>>
DN PREV200700526463
TI Aging of the mammalian circadian timing system: Changes in the central pacemaker and its regulation by photic and nonphotic signals.
AU Duncan, Marilyn J. [Reprint Author]
CS Univ Kentucky, Med Ctr, Dept Anat and Neurobiol, 800 Rose St, Lexington, KY 40536 USA
mjdunc0@uky.edu
SO Neuroembryology and Aging, (2006-2007) Vol. 4, No. 1-2, pp. 85-101. ISSN: 1661-3406. E-ISSN: 1661-3414.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 10 Oct 2007
Last Updated on STN: 10 Oct 2007
AB Aging alters many aspects of endogenously regulated, 24-hour (circadian) rhythms, such as their amplitude, relationship to the ambient lighting cycle, and sensitivity to phase resetting signals. In order to elucidate the mechanisms responsible for these age-related changes, many studies have investigated age-related changes in the neural components of the circadian timing system, which include the hypothalamic suprachiasmatic nucleus (SCN), the site of the mammalian master circadian pacemaker, its afferent projections from the retina, thalamus, and midbrain raphe, and its efferent projections to the hypothalamus, thalamus and limbic system. Studies have shown that the SCN exhibits age-related changes in electrical activity rhythms in the absence of neurodegeneration. Also, aging selectively decreases SCN expression of the circadian clock genes, Clock and Bmal1, as well as vasoactive intestinal peptide. The latter participates in mediating light-induced phase resetting of the circadian pacemaker and the amplitude of the preovulatory luteinizing hormone surge, which are both attenuated by aging. Furthermore, by decreasing serotonin 5-HT7 receptors in the dorsal raphe, aging reduces one type of nonphotic input to the SCN. This ***review*** describes which aspects of circadian rhythm ***regulation*** exhibit changes during ***aging*** , as well as which mechanisms appear to be spared or remain to be explored.

L8 ANSWER 4 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:517678 BIOSIS <<LOGINID::20080104>>
DN PREV200700517861
TI Sirtuins: critical regulators at the crossroads between cancer and aging.
AU Saunders, L. R.; Verdin, E. [Reprint Author]
CS Univ Calif San Francisco, Gladstone Inst Virol and Immunol, 1650 Owens St, San Francisco, CA 94158 USA
everdin@gladstone.ucsf.edu
SO Oncogene, (AUG 13 2007) Vol. 26, No. 37, pp. 5489-5504. CODEN: ONCNES. ISSN: 0950-9232.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 3 Oct 2007
Last Updated on STN: 3 Oct 2007
AB Sirtuins (SIRTs 1-7), or class III histone deacetylases (HDACs), are protein deacetylases/ADP ribosyltransferases that target a wide range of cellular proteins in the nucleus, cytoplasm, and mitochondria for post-translational modi. cation by acetylation (SIRT1, - 2, - 3 and - 5) or ADP ribosylation (SIRT4 and - 6). The orthologs of sirtuins in lower organisms play a critical role in ***regulating*** ***lifespan*** . As cancer is a disease of aging, we discuss the growing implications of the sirtuins in protecting against cancer development. Sirtuins regulate the cellular responses to stress and ensure that damaged DNA is not propagated and that mutations do not accumulate. SIRT1 also promotes replicative senescence under conditions of chronic stress. By participating in the stress response to genomic insults, sirtuins are thought to protect against cancer, but they are also emerging as direct participants in the growth of some cancers. Here, we ***review*** the growing implications of sirtuins both in cancer prevention and as specific and novel cancer therapeutic targets.

L8 ANSWER 5 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:437338 BIOSIS <<LOGINID::20080104>>
DN PREV200700441251
TI Interleukin-1 beta and caspase-1: Players in the ***regulation*** of ***age*** -related cognitive dysfunction.
AU Gemma, Carmelina; Bickford, Paula C. [Reprint Author]
CS Univ S Florida, Ctr Excellence Aging and Brain Repair, Coll Med, Dept

Neurosurg, 12901 Bruce B Downs Blvd,MDC-78, Tampa, FL 33612 USA
pbickfor@health.usf.edu
SO Reviews in the Neurosciences, (2007) Vol. 18, No. 2, pp. 137-148.
ISSN: 0334-1763.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 15 Aug 2007
Last Updated on STN: 15 Aug 2007
AB Scientific research on the unprecedented and growing number of older adults in the United States and other industrialized countries has focused much attention on the health consequences of aging. Over the last few decades, inflammation in the brain and its implication in the progression of aging and age-related cognitive dysfunction has been an area of increasing importance to neuroscientists and is now considered as one of the most interesting and promising topics for aging research. One of the critical aspects of inflammatory processes is that the activation of one upstream inflammatory molecule initiates a cascade of self-sustaining inflammatory events which leads to the activation of a number of different downstream functions. Recently, a great deal of attention has been given to the interplay between inflammatory and apoptotic processes and the regulation of these processes by the caspases. The caspase family of proteases can be divided into proapoptotic and pro-inflammatory members. The present ***review*** summarizes recent observations of the interactions between the inflammatory cytokine interleukin-1 (IL-1) beta and the inflammatory/apoptotic caspase-1 and their involvement in age-related impairments in cognition. A comprehensive understanding of these mechanisms could potentially lead to the development of preventive or protective therapies that reduce or inhibit the cognitive decline associated with aging and age-related neurodegenerative disease.

L8 ANSWER 6 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN
AN 2007:382202 BIOSIS <<LOGINID::20080104>>
DN PREV200700376724
TI Basic mechanisms of the aging gastrointestinal tract.
AU Salles, N. [Reprint Author]
CS Hosp Xavier Arnozan, Dept Geriatr, FR-33604 Pessac, France
nathalie.salles@chu-bordeaux.fr
SO Digestive Diseases, (2007) Vol. 25, No. 2, pp. 112-117.
ISSN: 0257-2753.
DT Article
LA English
ED Entered STN: 4 Jul 2007
Last Updated on STN: 4 Jul 2007
AB The goal of this short ***review*** is to summarize recent data on gastrointestinal changes with aging, focusing on gastrointestinal motility disorders, and mucosal variations. First of all, this ***review*** focused on gastrointestinal motility disorders with aging, even though an increased prevalence of several gastrointestinal motor disorders (i. e., dysphagia, dyspepsia, anorexia, and constipation) occurs in older people, aging per se appears to have a minor direct effect on most gastrointestinal functions. Secondly, this ***review*** focused on histological changes with ***aging*** , i. e., ***regulation*** of gastrointestinal mucosal growth, gastrointestinal carcinogenesis, and gastric mucosal changes, especially changes in gastric acid secretion, bacterial overgrowth and its consequences on elderly patients. Copyright (C) 2007 S. Karger AG, Basel.

L8 ANSWER 7 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN
AN 2007:290236 BIOSIS <<LOGINID::20080104>>
DN PREV200700283088
TI SIRT1 in transcription regulation.
AU Luo Lan [Reprint Author]; Gao Zheng-Nan; Cheng Li-Jing; Yang Ze
CS Beijing Hosp, Inst Geriatr, Beijing 100730, Peoples R China
rolland_304224@sohu.com; yangze016@yahoo.com.cn
SO Zhongguo Shengwu Huaxue yu Fenzi Shengwu Xuebao, (MAR 20 2007) Vol. 23,
No. 3, pp. 187-193.
ISSN: 1007-7626.
DT Article
General Review; (Literature Review)
LA Chinese
ED Entered STN: 2 May 2007
Last Updated on STN: 24 Oct 2007
AB The silent mating type information regulation 1 (SIRT1) is a recently discovered NAD-dependent protein deacetylase. The SIRT1 sequence has the closest homology to the S. cerevisiae Sir2 protein. Conservation of SIRT1 regulation of the insulin-insulin-like growth factor I signaling pathway has been observed for Caenorhabditis elegans and mammals, indicating an ancient role for SIRT1 in modulation of organism adaptations to nutritional intake. SIRT1 regulates gene expression in a variety of organism by deacetylation of modified lysine residues on histones, transcription factors and other proteins. The human SIRT1 regulates a number of transcription factors that modulate metabolism and endocrine signaling, including peroxisome proliferator-activated receptor gamma, peroxisome proliferator-activated receptor gamma coactivator 1 alpha. Therefore SIRT1 is characteristically involved in the regulation of several crucial cellular pathways including different signal pathways to ***regulate*** mammalian ***lifespan*** , gluconeogenesis and insulin secretion and so forth, the transcription regulator may play potential role in clinical application and modern medicine. In this ***review*** , the latest findings regarding SIRT1 gene and the role in regulation of transcription factors, mammalian lifespan and metabolism were reviewed and discussed.

L8 ANSWER 8 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN
AN 2007:161576 BIOSIS <<LOGINID::20080104>>
DN PREV200700160349
TI Advances in endocrinology of aging research, 2005-2006.
AU Bellino, Francis L. [Reprint Author]
CS 4899 Elmer Derr Rd, Frederick, MD 21703 USA
bellinof@mail.nih.gov
SO Experimental Gerontology, (DEC 2006) Vol. 41, No. 12, pp. 1228-1233.
CODEN: EXGEAB. ISSN: 0531-5565.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 7 Mar 2007
Last Updated on STN: 24 Oct 2007
AB The purpose of this brief ***review*** is to highlight some of the more important advances in endocrinology of aging research over the past year. Four advances were chosen and briefly described. First, exploration of the early steps in the generation of the internal steroidal hormonal signal involved in lifespan extension via the insulin/IGF-like signaling pathway in the nematode by two research groups revealed that the product of cholestanoic acid derivatives metabolized by a cytochrome P-450-like protein activates a protein with homology to the mammalian nuclear receptor superfamily, a process strikingly similar to the steroid hormone signaling pathway documented in mammalian systems. Second is the discovery that sirtuins, proteins that ***regulate*** ***lifespan*** in model organisms, enhance pancreatic insulin secretion in mice following a glucose challenge, suggesting the potential to ***regulate*** mammalian ***lifespan*** through ***regulation*** of the insulin signaling pathway. Third, the newly discovered hormone klotho, which also plays a role in ***regulating*** ***lifespan*** , in this case in mice, is reported to not only negatively affect insulin sensitivity but, perhaps more importantly, significantly affects calcium and phosphate metabolism as a required cofactor of Fgf-23 signaling. Finally the gonadotropin FSH is shown to directly affect bone density in mice separate from any direct effect of estrogen, suggesting that reproductive hormones other than estrogen can directly impact menopause-associated pathophysiology in non-reproductive tissues. (c) 2006 Elsevier Inc. All rights reserved.

L8 ANSWER 9 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN
AN 2007:114244 BIOSIS <<LOGINID::20080104>>
DN PREV200700112145
TI Vascular cell senescence - Contribution to atherosclerosis.
AU Minamino, Tohru; Komuro, Issei [Reprint Author]
CS Chiba Univ, Grad Sch Med, Dept Cardiovasc Sci and Med, Chuo Ku, 1-8-1 Inohana, Chiba 2608670, Japan
komuro-tky@umin.ac.jp
SO Circulation Research, (JAN 5 2007) Vol. 100, No. 1, pp. 15-26.
CODEN: CIRUAL. ISSN: 0009-7330.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 14 Feb 2007
Last Updated on STN: 14 Feb 2007
AB Cardiologists and most physicians believe that aging is an independent risk factor for human atherosclerosis, whereas atherosclerosis is thought to be a characteristic feature of aging in humans by many gerontologists. Because atherosclerosis is among the age-associated changes that almost always escape the influence of natural selection in humans, it might be reasonable to regard atherosclerosis as a feature of aging. Accordingly, when we investigate the pathogenesis of human atherosclerosis, it may be more important to answer the question of how we age than what specifically promotes atherosclerosis. Recently, genetic analyses using various animal models have identified molecules that are crucial for aging. These include components of the DNA-repair system, the tumor suppressor pathway, the telomere maintenance system, the insulin/Akt pathway, and other metabolic pathways. Interestingly, most of the molecules that influence the phenotypic changes of ***aging*** also ***regulate*** cellular senescence, suggesting a causative link between cellular senescence and aging. For example, DNA-repair defects can cause phenotypic changes that resemble premature aging, and senescent cells that show DNA damage accumulate in the elderly. Excessive calorie intake can cause diabetes and hyperinsulinemia, whereas dysregulation of the insulin pathway has been shown to induce cellular senescence in vitro. Calorie restriction or a reduction of insulin signals extends the lifespan of various species and decreases biomarkers of cellular senescence in vivo. There is emerging evidence that cellular senescence contributes to the pathogenesis of human atherosclerosis. Senescent vascular cells accumulate in human atheroma tissues and exhibit various features of dysfunction. In this ***review*** , we examine the hypothesis that cellular senescence might contribute to atherosclerosis, which is a characteristic of aging in humans.

L8 ANSWER 10 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN
AN 2007:80046 BIOSIS <<LOGINID::20080104>>
DN PREV200700068923
TI A stress response pathway involving sirtuins, forkheads and 14-3-3 proteins.
AU Berdichevsky, Ala; Guarente, Leonard [Reprint Author]
CS MIT, Dept Biol, 31 Ames St, Cambridge, MA 02139 USA
leng@mit.edu
SO Cell Cycle, (NOV 15 2006) Vol. 5, No. 22, pp. 2588-2591.
ISSN: 1538-4101.
DT Article
LA English
ED Entered STN: 24 Jan 2007
Last Updated on STN: 24 Jan 2007
AB A conserved sir2 deacetylase gene can determine longevity of yeast, flies and worms. Recently we have reported a molecular mechanism of action of the C. elegans homologue sir-2.1. Our study revealed a novel stress-dependent pathway for lifespan determination in which SIR-2.1 binds to 14-3-3 proteins and a forkhead transcription factor DAF-16 to activate transcription of DAF-16 target genes. DAF-16 has long been known as a central protein in the ***regulation*** of ***lifespan*** that interfaces with multiple pathways. Recent studies by us and other laboratories suggest that DAF-16 requires co-factors for full activity. In this prospective we ***review*** recent literature highlighting the role of SIR-2.1, 14-3-3 and other DAF-16 co-factors in DAF-16 activation.

=> d his

(FILE 'HOME' ENTERED AT 18:47:57 ON 04 JAN 2008)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 18:48:06 ON 04 JAN 2008
L1 4 S LBP 7 OR LBP7
L2 4 DUP REM L1 (0 DUPLICATES REMOVED)
L3 1 S T22G5.2
L4 2626198 S AGE OR AGING OR LIFESPAN
L5 149161 S L4 AND REGULA?
L6 13428 S L5 AND REVIEW
L7 5534 S L4 (3A) REGULAT?
L8 929 S L7 AND REVIEW

=> s l8 and lbp
L9 0 L8 AND LBP

=> s lbp and l4
L10 633 LBP AND L4

=> d bib abs

L10 ANSWER 1 OF 633 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN
AN 2007:607075 BIOSIS <<LOGINID::20080104>>
DN PREV200700612478
TI Fear avoidance beliefs do not influence disability and quality of life in Spanish elderly subjects with low back pain.
AU Kovacs, Francisco [Reprint Author]; Abraira, Victor; Cano, Alejandra; Royuela, Ana; Gil del Real, Maria Teresa; Gestoso, Mario; Mufraggi, Nicole; Muriel, Alfonso; Zamora, Javier
CS Fdn Kovacs, Dept Cientifico, Paseo Mallorca 36, E-07012 Palma de Mallorca, Spain
kovacs@kovacs.org
SO Spine, (SEP 1 2007) Vol. 32, No. 19, pp. 2133-2138.
CODEN: SPINDD. ISSN: 0362-2436.
DT Article
LA English
ED Entered STN: 6 Dec 2007
Last Updated on STN: 6 Dec 2007
AB Study Design. Correlation between previously validated questionnaires.Objective. To assess the association of fear avoidance beliefs (FAB) with disability and quality of life in elderly Spanish subjects.Summary of Background Data. As opposed to Anglo-Saxon and Northern European patients, in Spanish low back pain (***LBP***) patients of working ***age*** , the influence of FAB on disability and quality of life is sparse and much less than that of pain. The influence of FAB on ***LBP*** -related disability and quality of life in the elderly is unknown.Methods. A visual analogue scale (VAS), the Roland Morris Questionnaire (RMQ), the FAB-Phys questionnaire (FABQ), and the SF-12 questionnaire were used to assess ***LBP*** , disability, fear avoidance beliefs, and quality of life in 661 institutionalized elderly in Spain, 439 of whom had ***LBP*** .Results. In all subjects, FAB correlated with ***LBP*** (r = 0.477), disability (r = 0.458), the Physical Component Summary of SF-12 (PCS SF-12) (r = -0.389), and the Mental Component Summary of SF-12 (MCS SF-12) (r = -0.165). In subjects with ***LBP*** , FABs only correlated weakly with disability (r = -0.110). The stronger correlations were found between ***LBP*** and disability, both in all subjects (r = 0.803) and ***LBP*** patients (r = 0.445). Regression models including all the participants showed that the influence of FABs on physical quality of life was sparse and that FABs were not associated with either disability or mental quality of life. In elderly subjects with ***LBP*** , FABs were not associated with either disability or quality of life.Conclusion. In Spanish institutionalized elderly subjects, FABs only have a minor influence on physical quality of life, and none on disability or mental quality of life. In elderly subjects with ***LBP*** , differences in FABs are not associated with differences in disability or quality of life. Further studies should explore the potential value of FABs in the elderly in other settings.

=> s lipid binding protein and l4
L11 65 LIPID BINDING PROTEIN AND L4

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12 51 DUP REM L11 (14 DUPLICATES REMOVED)

=> d bib abs

L12 ANSWER 1 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:762489 CAPLUS <<LOGINID::20080104>>
DN 147:341248
TI Serum adipocyte fatty acid-binding protein levels were independently associated with carotid atherosclerosis
AU Yeung, D. C. Y.; Xu, A.; Cheung, C. W. S.; Wat, N. M. S.; Yau, M. H.; Fong, C. H. Y.; Chau, M. T.; Lam, K. S. L.
CS Department of Medicine and the Research Centre of Heart, Brain, Hormone, and Healthy Aging, Li Ka Shing Faculty of Medicine, Queen Mary Hospital, Univ. of Hong Kong, HKSAR, Peop. Rep. China
SO Arteriosclerosis, Thrombosis, and Vascular Biology (2007), 27(8), 1796-1802
CODEN: ATVBFA; ISSN: 1079-5642
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB Objective: Adipocyte fatty acid-binding protein (A-FABP) has been shown to be an important player in atherosclerosis in animal models. However, the clin. relevance of these findings is still unknown. This study aims to examine the relationship between serum A-FABP level and carotid intima-media thickness (IMT), an indicator of atherosclerosis in humans. Methods and Results- The study cohort included 479 Chinese subjects who underwent carotid IMT measurement. Serum A-FABP levels were detd. by enzyme-linked immunosorbent assays. Serum A-FABP levels pos. correlated with carotid IMT in both men (r = 0.211, P = 0.001) and women (r = 0.435, P < 0.001). In women, but not in men, the presence of plaques was assocd. with significantly higher serum A-FABP levels (P < 0.001 vs. women without plaques). Stepwise multiple regression anal. showed that serum A-FABP level was independently assocd. with carotid IMT in women (P = 0.034), together with ***age*** and hypertension (both P < 0.001). Conclusions: A-FABP is an independent determinant of carotid atherosclerosis in Chinese women, but not in men. This gender difference may be attributed to the lower serum A-FABP levels in men, and the effect of other risk factors, such as smoking, among our male participants. Our results have provided clin. evidence supporting the role of A-FABP in the development of atherosclerosis.
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d bib abs 2-10

L12 ANSWER 2 OF 51 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:442130 BIOSIS <<LOGINID::20080104>>
DN PREV200700428784
TI Reduced annexin II protein expression in high-grade prostatic intraepithelial neoplasia and prostate cancer.
AU Yee, David S.; Narula, Navneet; Ramzy, Ibrahim; Boker, John; Ahlering, Thomas E.; Skarecky, Douglas W.; Ornstein, David K. [Reprint Author]
CS Univ Calif Irvine, Dept Urol, Med Ctr, 101 The City Dr,Bldg 55,Room 300,Rte 81, Orange, CA 92868 USA
dornstei@uci.edu
SO Archives of Pathology & Laboratory Medicine, (JUN 2007) Vol. 131, No. 6, pp. 902-908.
CODEN: APLMAS. ISSN: 0003-9985.
DT Article
LA English
ED Entered STN: 15 Aug 2007
Last Updated on STN: 15 Aug 2007
AB Context. - Annexin II is a calcium-dependent phospho- ***lipid*** - ***binding*** ***protein*** that plays a role in many cellular functions, including apoptosis, signal transduction, and cellular motility. The protein is strongly expressed in normal prostatic epithelial glands, but its expression in benign prostatic lesions has not been reported. Although commonly underexpressed in prostate cancer, the association of reduced expression with pathologic grade and stage is unknown.Objective. - To compare annexin II expression in benign prostatic lesions with expression in high-grade prostatic intraepithelial neoplasia and prostate cancer, as well as to correlate expression levels with pathologic grade and stage.Design. - A semi-quantitative assessment of annexin II expression was performed in radical prostatectomy specimens from 74 patients and prostate needle core biopsy specimens from 13 patients. Foci with normal prostatic glands, atrophic glands, basal cell hyperplasia, high-grade prostatic intraepithelial neoplasia, and prostatic

adenocarcinoma were evaluated.Results. - Annexin II expression was present in more than 50% of glands in most (>85%) samples of benign prostatic epithelium, atrophic glands, and basal cell hyperplasia. In high-grade prostatic intraepithelial neoplasia, annexin II staining was markedly reduced in epithelial cells but not in basal cells. Annexin II was absent or focally present in moderately differentiated adenocarcinoma but was retained in poorly differentiated adenocarcinomas.Conclusions. - Reduced annexin II expression may be a useful diagnostic biomarker to help identify small foci of moderately differentiated adenocarcinoma on needle core biopsy specimens since it is consistently expressed in benign prostatic glands. Re-expression of annexin II in poorly differentiated adenocarcinoma may provide prognostic information.

L12 ANSWER 3 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:966059 CAPLUS <<LOGINID::20080104>>
DN 147:424776
TI Adipocyte fatty acid-binding protein is associated with markers of obesity, but is an unlikely link between obesity, insulin resistance, and hyperandrogenism in polycystic ovary syndrome women
AU Moehlig, Matthias; Weickert, Martin O.; Ghadamgadai, Elham; Machlitt, Andrea; Pfueller, Bettina; Arafat, M.; Pfeiffer, Andreas F. H.; Schoefl, Christof
CS Department of Endocrinology, Diabetes and Nutrition, Charite-University Medicine Berlin, Berlin, 12200, Germany
SO European Journal of Endocrinology (2007), 157(2), 195-200
CODEN: EJOEEP; ISSN: 0804-4643
PB BioScientifica Ltd.
DT Journal
LA English
AB Objective: Many polycystic ovary syndrome (PCOS) women suffer from adiposity and insulin resistance (IR), which play an important role in the development and maintenance of PCOS. Adipocyte fatty acid-binding protein (A-FABP) is mainly expressed in adipocytes, and circulating A-FABP has been assocd. with markers of obesity and IR. Thus, as obsd. with other adipose tissue derived factors, secreted A-FABP might be involved in the pathogenesis of obesity-assocd. disorders such as PCOS. Design: Plasma A-FABP concns. were measured in 102 non-diabetic PCOS women, and assocns.
with markers of obesity. IR, inflammation, and hyperandrogenism were investigated by correlation and multiple linear regression analyses. The effect of lifestyle intervention on A-FABP was studied in a second cohort of 17 obese PCOS women. Results: A-FABP correlated with body mass index (BMI; R = 0.694, P<0.001), dual-energy x-ray-absorptiometry (DEXA) fat mass (R = 0.729, P<0.001), DEXA lean body mass (R = 0.399, P = 0.001), HOMA %S (R = -0.435, P<0.001), hsCRP (R = 0.355, P = 0.001), and free testosterone (fT; R = 0.230, P = 0.02). Adjusted for ***age*** , smoking, and glucose metab. the assocn. of A-FABP with HOMA %S was still significant (P<0.001), whereas the assocns. with fT (P = 0.09) and hsCRP (P = 0.25) were not. Inclusion of BMI into the model abolished the impact of A-FABP on HOMA %S. In BMI-matched PCOS women (n = 20 pairs), neither HOMA %S (P = 0.3) nor fT (P = 0.6) were different despite different A-FABP levels (P<0.001), and in 17 obese PCOS women undergoing a lifestyle intervention, changes in IR were not paralleled by changes in A-FABP. Conclusions: Circulating A-FABP was correlated with markers of obesity, but had no major impact on IR, inflammation, or hyperandrogenemia in PCOS women.
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:67089 CAPLUS <<LOGINID::20080104>>
DN 146:355212
TI Genetic variability affects the development of brown adipocytes in white fat but not in interscapular brown fat
AU Xue, Bingzhong; Rim, Jong-Seop; Hogan, Jessica C.; Coulter, Ann A.; Koza, Robert A.; Kozak, Leslie P.
CS Pennington Biomedical Research Center, Baton Rouge, LA, 70808, USA
SO Journal of Lipid Research (2007), 48(1), 41-51
CODEN: JLPRAW; ISSN: 0022-2275
PB American Society for Biochemistry and Molecular Biology, Inc.
DT Journal
LA English
AB Cold exposure induces brown adipocytes in retroperitoneal fat (RP) of adult A/J mice but not in C57BL/6J (B6) mice. In contrast, induction of the mitochondrial uncoupling protein 1 gene (Ucp1) in interscapular brown adipose tissue (iBAT) shows no strain dependence. We now show that unlike iBAT, in which Ucp1 was expressed in the fetus and continued throughout life, in RP, Ucp1 was transiently expressed between 10 and 30 days of ***age*** and then disappeared. Similar to the lack of genetic variation in the expression of Ucp1 in iBAT during cold induction of adult mice, no genetic variation in Ucp1 expression in iBAT was detected during development. In contrast, UCP1-pos. multilocular adipocytes, together with corresponding increases in Ucp1 expression, appeared in RP at 10 days of ***age*** in A/J and B6 mice, but with much higher expression in A/J mice. At 20 days of ***age*** , brown adipocytes represent the major adipocyte present in RP of A/J mice. The disappearance of brown adipocytes by 30 days of ***age*** suggested that tissue remodeling occurred in RP. Genetic variability in Ucp1 expression could not be explained by variation in the expression of selective transcription factors and signaling mols. of adipogenesis. In summary, the existence of genetic variability between A/J and B6 mice during the development of brown adipocyte expression in RP, but not in iBAT, suggests that developmental mechanisms for the brown adipocyte differentiation program are different in these adipose tissues.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:830502 CAPLUS <<LOGINID::20080104>>
DN 147:423577
TI Adipocyte- and Heart-Type Fatty Acid Binding Proteins Are Both Expressed in Subcutaneous and Intramuscular Porcine (Sus scrofa) Adipocytes
AU Gardan, Delphine; Louveau, Isabelle; Gondret, Florence
CS INRA, UMR1079 Systemes d'Elevage Nutrition Animale et Humaine, Saint Gilles, F-35590, Fr.
SO Comparative Biochemistry and Physiology, Part B: Biochemistry & Molecular Biology (2007), 148B(1), 14-19
CODEN: CBPBB8; ISSN: 1096-4959
PB Elsevier B.V.
DT Journal
LA English
AB Adipocyte- (A) and heart- (H) type fatty acid binding proteins (FABP) contribute to efficient fat storage and utilization, resp. To understand regional-differences in lipid metab. between tissues, A- and H-FABP transcript and protein levels were studied in adipocytes isolated from s.c. adipose tissue or skeletal muscle in growing pigs (Sus scrofa). Interestingly, H-FABP was expressed in adipocytes isolated from both sites. .We also showed that A-FABP and H-FABP were expressed at a lower level in i.m. adipocytes than in s.c. adipocytes. A discrepancy was obsd. between ***age*** -related changes in A-FABP content in isolated adipocytes and cell diam. or lipid content variations in tissues during growth.
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:1012780 CAPLUS <<LOGINID::20080104>>
DN 145:349629
TI Glomerular and podocyte-specific expression profiling in mouse kidney and construction of the GlomChip microarray
IN Betsholtz, Christer; Tryggvason, Karl; Takemoto, Minoru; He, Liqun; Patrakkas, Jaakko
PA Swed.
SO PCT Int. Appl., 802pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006100066	A1	20060928	WO 2006-EP2646	20060322
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006216722	A1	20060928	US 2005-90997	20050325
PRAI US 2005-90997	A	20050325		

AB The present invention provides the assembly and use of a transcription-profiling platform dedicated to the study of mouse renal glomeruli. By using a novel method for glomerulus isolation, a series of high complexity EST libraries were constructed from glomeruli at different stages of development. From these libraries, a total of 15,627 EST clones were sequenced, and by annotation against ENSEMBL found to map to 6,053 different genes, estd. to cover 85% of the glomerular transcriptome. Microarray anal. of isolated glomeruli, non-glomerular kidney tissue, isolated extra-renal microvessel fragments, and FACS-sorted podocytes identified most known glomerular and podocyte-specific transcripts. To identify novel podocyte-specific transcripts, the EST clones were arrayed and hybridized against labeled targets from isolated glomeruli, non-glomerular kidney tissue, FACS-sorted podocytes, and brain capillary fragments. This revealed the existence of over 300 novel glomerular cell-enriched transcripts, the expression of many of which was further localized to podocytes, mesangial cells, and juxtaglomerular cells by in situ hybridization. For one of the podocyte-restricted transcripts, dendrin, previously regarded to be brain-specific, the protein was expressed, antibodies generated, and the antibodies used to localize dendrin to the podocyte foot processes. Quant. expression data are provided for known podocyte genes. Construction and performance of a glomerular transcript-specific microarray, designated GlomChip, is described.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:33751 CAPLUS <<LOGINID::20080104>>
DN 144:128966
TI Constrained cyano compounds as selective inhibitors of dipeptidyl peptidase IV, their preparation, pharmaceutical compositions, and use in therapy
IN Campbell, David Alan; Betancort, Juan Manuel; Winn, David T.
PA USA
SO U.S. Pat. Appl. Publ., 35 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2006009518	A1	20060112	US 2005-179797	20050712
CA 2573848	A1	20060216	CA 2005-2573848	20050712
WO 2006017292	A1	20060216	WO 2005-US24695	20050712

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

EP 1778220	A1	20070502	EP 2005-773420	20050712

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR

PRAI US 2004-587391P P 20040712
WO 2005-US24695 W 20050712
OS CASREACT 144:128966; MARPAT 144:128966
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The invention relates to constrained cyano compds. of formula I, which are selective inhibitors of dipeptidyl peptidase IV (DPP-IV). In compds. I, X is (un)substituted C, optionally forming a double bond with one of the carbon atoms to which it is attached, S, or O; R1 and R4 are independently H, (un)substituted alkyl, (un)substituted alkenyl, (un)substituted alkynyl, (un)substituted cycloalkyl, (un)substituted cycloalkyl-alkyl, (un)substituted aryl, (un)substituted aralkyl, etc.; and R2, R3, R5, and R6 are independently selected from H, F, Cl, Br, I, OH, NH2, CN, alkoxy, (di)alkylamino, acyl, alkoxycarbonyl, aryloxy, etc. The invention also relates to the prepn. of I, pharmaceutical compns. comprising a compd. I together with at least one pharmaceutically acceptable carrier or diluent, optionally in combination with another active ingredient, as well as to the use of the compns. for treating, controlling, or preventing conditions affected by dipeptidyl peptidase-IV inhibition. Esterification of (S)-phenylglycine followed by condensation with benzaldehyde, .alpha.-allylation, hydrolysis and N-protection gave amino acid II, which underwent ozonolysis, ester hydrolysis, and cyclization with L-cysteine Me ester to give thiazolidine III. Intramol. cyclocondensation of III, amidation, dehydration and deprotection resulted in the formation of hexahydropyrrolothiazole IV. The compds. of the invention are selective for DPP-IV over other dipeptidyl peptidases with compd. IV being more than 100-fold selective for DPP-IV over DPP-VII, DPP-VIII, and fibroblast activation protein (FAP) and between 10- and 100-fold for DPP-IV over DPP-IX.

L12 ANSWER 8 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:741512 CAPLUS <<LOGINID::20080104>>
DN 145:245635
TI Klotho protein promotes adipocyte differentiation
AU Chihara, Yukana; Rakugi, Hiromi; Ishikawa, Kazuhiko; Ikushima, Masashi; Maekawa, Yoshihiro; Ohta, Junsuke; Kida, Iwao; Ogihara, Toshio
CS Department of Geriatric Medicine, Osaka University Graduate School of Medicine, Osaka, 565-0871, Japan
SO Endocrinology (2006), 147(8), 3835-3842
CODEN: ENDOAO; ISSN: 0013-7227
PB Endocrine Society
DT Journal
LA English
AB Mice with homozygous disruption of the klotho exhibit multiple ***age*** -related disorders and have barely detectable amts. of white adipose tissue. Although klotho expression in cultured adipocytes has been reported, little is known about its function in adipocytes. In the present study, we investigated the role of klotho on adipocyte differentiation. Adipocyte differentiation was induced by incubation of confluent 3T3-L1 cells with insulin, dexamethasone, and 1-methyl-3-isobutyl-xanthin. Klotho-siRNA and expression vector were produced for klotho suppression and overexpression, resp. Klotho protein was purified for detn. of the hormonal effect of klotho. Klotho mRNA and protein expression increased up to the 3rd d of differentiation. A peroxisome proliferator-activated receptor-.gamma. agonist increased klotho expression during the early period of adipocyte differentiation. The mRNA expression of adipocyte differentiation markers, such as CCAAT/enhancer-binding protein (C/EBP).alpha., C/EBP.beta., C/EBP.delta., peroxisome proliferator-activated receptor-.gamma., and fatty acid binding protein 4, was decreased by klotho suppression, and increased 1.9- to 3.8-fold by klotho overexpression. The results of Oil Red O staining also suggested that klotho overexpression promoted adipocyte differentiation. Klotho protein stimulation resulted in a 2.4- to 4.6-fold increase in mRNA expression of differentiation markers compared with control, and the time course depended on adipocyte induction status. Western blot anal. showed that protein levels of C/EBP.alpha. and C/EBP.delta. were increased by klotho protein stimulation. These results suggest that klotho works as a hormonal factor to promote adipocyte differentiation in the early days, during the period of transient proliferation in the differentiation process, and that klotho may play an essential role in adipocyte differentiation.
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 9 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:1070578 CAPLUS <<LOGINID::20080104>>
DN 145:349038
TI The farnesoid X receptor promotes adipocyte differentiation and regulates adipose cell function in vivo
AU Rizzo, Giovanni; Disante, Moises; Mencarelli, Andrea; Renga, Barbara; Gioiello, Antimo; Pellicciari, Roberto; Fiorucci, Stefano
CS Dipartimento di Medicina Clinica e Sperimentale, University of Perugia, Perugia, Italy
SO Molecular Pharmacology (2006), 70(4), 1164-1173
CODEN: MOPMA3; ISSN: 0026-895X
PB American Society for Pharmacology and Experimental Therapeutics
DT Journal
LA English
AB The differentiation of a preadipocyte into a mature adipocyte is a highly regulated process that requires a scripted program of transcriptional events leading to changes in gene expression. Several genes are assocd. with adipogenesis, including the CAAT/enhancer-binding protein (C/EBPs) and peroxisome proliferator-activated receptor (PPAR) families of transcription factors. In this study, we have investigated the role of the farnesoid X receptor (FXR), a bile acid-activated nuclear receptor, in regulating adipogenesis in a preadipocyte cell line (3T3-L1 cells). Our results show that FXR is expressed in the white adipose tissue of adult mice and in differentiated 3T3-L1 cells but not in undifferentiated preadipocytes. Exposure of 3T3-L1 cells to INT-747 (6-Et cheno-deoxycholic acid), a potent and selective FXR ligand, increases preadipocyte differentiation induced by a differentiating mixt. contg. insulin. Augmentation of differentiating mixt.-induced differentiation of 3T3-L1 cells by INT-747 assocd. with induction of aP2, C/EBP.alpha., and PPAR.gamma.2 mRNAs along with other adipocyte-related genes. This effect was reversed by guggulsterone, an FXR antagonist, and partially reverted by GW9662 (2-chloro-5-nitro-N-phenylbenzamide), a selective PPAR.gamma. antagonist, indicating that FXR modulates adipocyte-related genes by PPAR.gamma.-dependent and -independent pathways. Regulation of adipocyte-related genes by INT-747 was lost in FXR-/- mice, indicating that modulation of these genes by INT-747 requires an intact FXR. In addn., INT-747 enhances both insulin-induced serine phosphorylation of Akt and glucose uptake by 3T3-L1 cells. Taken together, these results suggest that activation of FXR plays a crit. role in regulating adipogenesis and insulin signaling.
RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 10 OF 51 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
AN 2006598155 EMBASE <<LOGINID::20080104>>
TI Localization of LRRK2 to membranous and vesicular structures in mammalian brain.
AU Biskup S.; Moore D.J.; Celsi F.; Higashi S.; West A.B.; Andrabi S.A.; Kurkinen K.; Yu S.-W.; Savitt J.M.; Waldvogel H.J.; Faull R.L.M.; Emson P.C.; Torp R.; Ottersen O.P.; Dawson T.M.; Dawson V.L.
CS Dr. V.L. Dawson, Johns Hopkins University, School of Medicine, Broadway Research Building, 733 North Broadway, Baltimore, MD 21205, United States. vdawson@jhmi.edu
SO Annals of Neurology, (Nov 2006) Vol. 60, No. 5, pp. 557-569.
Refs: 23
ISSN: 0364-5134 CODEN: ANNED3
CY United States
DT Journal; Article
FS 029 Clinical and Experimental Biochemistry
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 4 Jan 2007
Last Updated on STN: 4 Jan 2007
AB Objective: The PARK8 gene responsible for late-onset autosomal dominant Parkinson's disease encodes a large novel protein of unknown biological function termed leucine-rich repeat kinase 2 (LRRK2). The studies herein explore the localization of LRRK2 in the mammalian brain. Methods: Polyclonal antibodies generated against the amino or carboxy termini of LRRK2 were used to examine the biochemical, subcellular, and

immunohistochemical distribution of LRRK2. Results: LRRK2 is detected in rat brain as an approximate 280kDa protein by Western blot analysis. Subcellular fractionation demonstrates the presence of LRRK2 in microsomal, synaptic vesicle-enriched and synaptosomal cytosolic fractions from rat brain, as well as the mitochondrial outer membrane. Immunohistochemical analysis of rat and human brain tissue and primary rat cortical neurons, with LRRK2-specific antibodies, shows widespread neuronal-specific labeling localized exclusively to punctate structures within perikarya, dendrites, and axons. Confocal colocalization analysis of primary cortical neurons shows partial yet significant overlap of LRRK2 immunoreactivity with markers specific for mitochondria and lysosomes. Furthermore, ultrastructural analysis in rodent basal ganglia detects LRRK2 immunoreactivity associated with membranous and vesicular intracellular structures, including lysosomes, endosomes, transport vesicles, and mitochondria. Interpretation: The association of LRRK2 with a variety of membrane and vesicular structures, membrane-bound organelles, and microtubules suggests an affinity of LRRK2 for lipids or lipid-associated proteins and may suggest a potential role in the biogenesis and/or regulation of vesicular and membranous intracellular structures within the mammalian brain. .COPYRGT. 2006 American Neurological Association Published by Wiley-Liss, Inc., through Wiley Subscription Services.

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L13 3 A FABP AND (LIFESPAN OR AGING)

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YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:85781 CAPLUS <<LOGINID::20080104>>
DN 146:180307
TI Method for the determination of adipocyte fatty acid binding protein (***A*** - ***FABP*** , FABP4, P2) and diagnosis of metabolic syndrome, diabetes type II and insulin resistance
PA Biovendor Laboratory Medicine, Inc., Czech Rep.
SO Ger. Offen., 36pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 102006034607	A1	20070125	DE 2006-102006034607	20060721
WO 2007063363	A2	20070607	WO 2006-IB2383	20060721
WO 2007063363	A3	20070830		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRAI DE 2005-102005034788 IA 20050721
AB The invention concerns a method for diagnosing metabolic syndrome, its early forms and complications, diabetes type II, insulin resistance, obesity or similar diseases by detg. the concn. of adipocyte fatty acid binding protein ***A*** - ***FABP*** in a body fluid and using the result as an indicator of the fatty acid transport capacity. ***A*** - ***FABP*** is detd. by ELISA or similar dye-involving immunoassays in conjunction with microtiterplates; recombinant ***A*** - ***FABP*** is used as ref. material. For diagnosis addnl. data are taken into account, e.g. body mass index, glucose level.

L14 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:830502 CAPLUS <<LOGINID::20080104>>
DN 147:423577
TI Adipocyte- and Heart-Type Fatty Acid Binding Proteins Are Both Expressed in Subcutaneous and Intramuscular Porcine (Sus scrofa) Adipocytes
AU Gardan, Delphine; Louveau, Isabelle; Gondret, Florence
CS INRA, UMR1079 Systemes d'Elevage Nutrition Animale et Humaine, Saint Gilles, F-35590, Fr.
SO Comparative Biochemistry and Physiology, Part B: Biochemistry & Molecular Biology (2007), 148B(1), 14-19
CODEN: CBPBB8; ISSN: 1096-4959
PB Elsevier B.V.
DT Journal
LA English
AB Adipocyte- (A) and heart- (H) type fatty acid binding proteins (FABP) contribute to efficient fat storage and utilization, resp. To understand regional-differences in lipid metab. between tissues, A- and H-FABP transcript and protein levels were studied in adipocytes isolated from s.c. adipose tissue or skeletal muscle in growing pigs (Sus scrofa). Interestingly, H-FABP was expressed in adipocytes isolated from both sites. We also showed that ***A*** - ***FABP*** and H-FABP were expressed at a lower level in i.m. adipocytes than in s.c. adipocytes. A discrepancy was obsd. between age-related changes in ***A*** - ***FABP*** content in isolated adipocytes and cell diam. or lipid content variations in tissues during growth.
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:385929 CAPLUS <<LOGINID::20080104>>
DN 141:52311
TI Genetic polymorphisms affecting the phenotypic expression of familial hypercholesterolemia
AU Bertolini, Stefano; Pisciotta, Livia; Di Scala, Lilla; Langheim, Silvia; Bellocchio, Antonella; Masturzo, Paola; Cantafora, Alfredo; Martini, Scipione; Averna, Maurizio; Pes, Gianni; Stefanutti, Claudio; Calandra, Sebastiano
CS Department of Internal Medicine, University of Genoa, Genoa, I-16132, Italy
SO Atherosclerosis (Amsterdam, Netherlands) (2004), 174(1), 57-65
CODEN: ATHSBL; ISSN: 0021-9150
PB Elsevier
DT Journal
LA English
AB The clin. expression of heterozygous familial hypercholesterolemia (FH) is highly variable even in patients carrying the same LDL receptor (LDL-R) gene mutation. This variability might be due to environmental factors as well as to modifying genes affecting lipoprotein metab. We investigated Apo E (.epsilon.2, .epsilon.3, .epsilon.4), MTP (-493G/T), Apo B (-516C/T), Apo A-V (-1131T/C), HL (-514C/T and -250G/ ***A***), ***FABP*** -2 (A54T), LPL (D9N, N291S, S447X) and ABCA1 (R219K) polymorphisms in 221 unrelated FH index cases and 349 FH relatives with defined LDL-R gene mutations. We found a significant and independent effect of the following polymorphisms on: (i) plasma LDL-C (Apo E, MTP and Apo B); (ii) plasma HDL-C (HL, FABP-2 and LPL S447X); (iii) plasma triglycerides (Apo E and Apo A-V). In subjects with coronary artery disease (CAD+), the prevalence of FABP-2 54TT genotype was higher (16.5% vs. 5.2%) and that of ABCA1 219RK and KK genotypes lower (33.0% vs. 51.5%) than in subjects with no CAD. Independent predictors of increased risk of CAD were male sex, age, arterial hypertension, LDL-C level and FABP-2 54TT genotype, and of decreased risk the 219RK and KK genotypes of ABCA1. These findings show that several common genetic variants influence the lipid phenotype and the CAD risk in FH heterozygotes.
RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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FULL ESTIMATED COST		162.23	162.44

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) TOTAL	ENTRY	SINCE FILE SESSION	
CA SUBSCRIBER PRICE		-10.40	-10.40

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